THE SUDDEN ONSET OF PARALYSIS IN POTT'S DISEASE WITHOUT DEFORMITY OF THE VERTEBRÆ.

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Compression of the spinal cord causing paralysis may be due to various morbid processes, but here I shall refer only to compression following tuberculous caries of the vertebræ or Pott's disease. Although both the clinical and pathological manifestations of this disease have been carefully studied, there are still many disputed points in regard to the exact means by which interruption of the functions of the spinal cord are brought about, so for this reason, and on account of some very unusual features the following cases seemed worthy of careful study. I wish particularly to express my thanks to Dr. Spiller, Dr. Mills, and Dr. Frazier who have kindly furnished me with the material upon which this investigation is based.

Case I.—A colored male, aged thirty-eight years, was admitted to the Philadelphia Hospital in the service of Dr. C. K. Mills, November 7, 1904, complaining of paraplegia. His family history revealed nothing of importance. He denied syphilis. On August 7, 1904, he became very constipated, not having had a bowel movement for fourteen days; at the same time he had considerable abdominal pain. After evacuation of the bowels by means of purgatives, the pain subsided but still lingered. About one month later his right leg suddenly gave way while walking, associated with cramp-like pain. The pain persisted two days, and then the right leg became totally paralyzed. One week later the left leg was similarly affected but without pain. Again he became very constipated and had some difficulty in urinating. Since that time the patient has been confined to bed, and he says that he has not been able to feel anything in either lower extremity. Occasionally he had girdle sensation.

The following notes were made on the day of admission: The patient is fairly well developed. He can wrinkle his forehead evenly on both sides. The nasolabial folds are of equal prominence, he can show teeth, whistle, and draw mouth back on either side. The masseter and temporal muscles contract promptly. There seems to be some wasting of the face but it is symmetrical. Both pupils are round and equal and react promptly to light and accommodation. Sensation for touch and pain is normal over the face and neck. The extra-ocular muscles are normal. Motion and resistance to passive movements and grasp of hands is good on both sides. There is very slight ataxia of both upper limbs as shown by the finger to nose test. Sense of position is normal on both sides and sensation for touch, pain, heat, and cold is normal in the upper limbs. The lungs are hyper-resonant anteriorly and harsh breath sounds are heard over the upper portions. No rales can be elicited. The heart is normal. The abdomen is somewhat distended but not tympanitic. Palpation elicits no pain or tenderness. The abdominal organs seem normal. Sensation for touch and pain is normal over chest and abdomen; sensation for heat and cold is normal down to a point two inches below the umbilicus but below this level thermal sense is lost. There is marked atrophy of both lower limbs. Motion and resistance to passive movements is nil in both limbs. Patient is unable to move even toes. There is double foot drop. Impossible to test for ataxia on account of paralysis. Both limbs are slightly spastic, the left more so. At intervals there are involuntary contractions of the flexor and adductor muscles of each thigh. The right knee jerk is exaggerated, the left seems normal or slightly decreased. Ankle clonus is present on both sides, but it is much more persistent on the right. Babinski reflex is distinct on both sides. Sensation to touch is normal, on the left limb except for space extending downward six inches from the middle of the leg from that point downward it seems to be diminished, on the right side it is present, but the man is unable to locate touch on leg. Sensation to pain is present on the right limb but is diminished and he is unable to locate pin pricks. Sensation to pain on the left side is about the same as on the right. Sensation to heat and cold is absolutely lost over the whole of both lower extremities. Achilles jerk is absent on the right, but present on the left.

Two days later a slight angular deformity was discovered corresponding to the ninth thoracic vertebra. On November 22, all forms of sensation including touch, pain, and temperature were lost over both lower extremities and extended upward as far as the level of the umbilicus. A large bed sore had developed over the sacral region. The patient died November 30. The autopsy was performed the next morning.

Anatomical Diagnosis. Bilateral pleural adhesions, obliterative pericarditis, chronic fibrous tuberculosis of the lungs, malposition of the left kidney, gastrectasis, and enteroptosis, miliary tuberculosis of liver, dilatation and ulceration of œsophagus, subphrenic abscess, caries of lower thoracic vertebræ, tuberculous external pachymeningitis, internal hydrocephalus.

Microscopic examination of the lower thoracic segments where the pressure was greatest showed that the outline of the cord was not altered. The pia was slightly thickened on the anterior aspect, the adventitia of the pial vessels was less compact than normal and was infiltrated with small round cells. Here and there the pia was separated from the cord, the spaces between containing a few round cells and red blood corpuscles. In the white matter the neuroglia cells were swollen and increased in number; and the glia septa were thickened, standing out prominently. Many granular cells and a few round cells were seen diffusely scattered. The white matter presented the characteristic vesicular appearance. The medullary sheaths were distended, many axis cylinders had entirely disappeared and others were swollen or atrophied. The bloodvessels were thickened and there was moderate perivascular round-cell infiltration. Although the white substance was severely diseased the area of the lateral pyramidal tracts was perhaps more affected than the other parts. The central portions of the gray matter were entirely destroyed, including all of the gray commissures and part of the anterior horns, leaving a large irregular cavity with ragged edges containing debris, few small round cells, red blood corpuscles and compound granular cells. In the remaining portions of the gray matter there were scattered round cells and an occasional granular cell. Here the bloodvessels were also thickened, and there was moderate perivascular infiltration. The necrotic cavity did not extend beyond the limits of the eighth dorsal segment but the central canal in the adjoining sections above and below was distended and the ependyma cells were partially destroyed.

In the upper dorsal, cervical, and lower lumbar segments there was some thickening of the bloodvessels within the gray matter and slight perivascular infiltration. This was more marked in the branches of the anterior spinal artery near the base of the anterior horns. With the Weigert method there was degeneration of Goll's column extending upward into the cervical region. In the lower lumbar segments no distinct degenerations were found. At the level of compression many ganglion cells within the gray matter had been destroyed but a few remained which appear swollen, and with the Weigert stain the nuclei were indistinct. The Nissl stain could not be used. The bloodvessels of the pia about the pons and medulla seemed somewhat thicker than normal and some showed slight perivascular infiltration. The cells of the choroid plexus about the fourth ventricle were very much swollen and the bloodvessels were thickened. Sections of the cerebral cortex presented some rather doubtful round cell infiltration of the pia.

CASE II.—J. D., was admitted July 16, 1904, to the service of Dr. Spiller, complaining of paralysis of both lower extremities. Three years previously his left leg had been amputated six inches below the knee as a result of injury. He denied syphilis. His family history was negative. July 2, 1904, he was compelled to go to bed on account of severe dull aching pain just below the angle of the scapula. Two days later there was much numbness and tingling just below the painful areas and on that day he suddenly lost power in both lower extremities. During this time he had had retention of urine and was constipated.

The following notes were dictated two days after his admission: The patient is fairly well nourished. Pupils are round and equal and react promptly to light and accommodation. The extraocular muscles are normal. Facial muscles contract equally and promptly; movements of upper extremities are equal and normal. The epigastric and abdominal reflexes are absent on both sides. Cremasteric reflex is absent on the left and diminished on the right. The knee jerks and Achilles jerks are absent on both sides. There is no ankle clonus on either side. Sensation for pain and touch is lost over both legs and abdomen up to a point two and one-half inches below the right nipple, and about one inch lower down on the left side. Adjoining this line above is a zone of hyperesthesia about two inches wide, which narrows to about one inch in width near the axilla of either side. Testicular pain is absent.

The following additional notes were dictated by Dr. Spiller some days later: Sensation for touch, pain, and temperature is similar to the above. Cremasteric reflex is present on both sides but diminished. Passive movement of the stump of left leg on thigh causes involuntary jerking movement of this limb of which the patient is not conscious without seeing it, but he is conscious of a jarring sensation in his back. These involuntary movements are not distinct in the right lower limb. He has no knowledge of the sense of position of his legs. The knee jerks are both absent, the plantar reflex is present on the left side, but sluggish on the right. There is an area of tenderness of the spinous processes about the level of the spines of the scapula, but no deformity.

The patient died July 28, 1904.

Macroscopic examination of the spinal cord and its membranes showed an external pachymeningitis extending from the lower cervical region to the upper lumbar. There was a considerable amount of necrotic cheesy material which by microscopic examination revealed the characteristic picture of tuberculous disease. Microscopic examination of sections taken from the upper thoracic portion of the spinal cord showed no diminution in its volume. When stained by the hemalum and fuchsin method there was slight cellular infiltration of the pia, and the pial vessels presented slight infiltration of the adventitia. In the white substance there were numerous vacuoles, mononuclear cellular infiltration, thickening of the bloodvessels and slight perivascular infiltration. The neuroglia cells were increased and here and there were a few granular cells. The anterior portions of the posterior columns were partially destroyed. The anterior longitudinal septum was infiltrated with round cells and appeared more prominent than normal. In the gray substance there was moderate diffuse cellular infiltration of the mononuclear type and red blood cells as well as compound granular cells were found. The blood vessels which were cut in cross section appeared thickened, and the perivascular and perigangliar spaces were enlarged. The most marked inflammatory changes were found about the gray commissures and central portions of the anterior horns. In some sections necrosis has taken place in the area supplied by the commissural branch of the anterior spinal artery. With the Weigert method the posterior and lateral columns suffered most, and the anterior columns least. Axis cylinders had disappeared and many remaining were swollen, while here and there a few normal fibers were seen. Many of the ganglion cells of the anterior horns had disappeared, others stained poorly, and their nuclei were indistinct. In the lower thoracic lumbar and cervical segments the process was similar in the gray matter, but much less marked, while the white substance was little affected. Distinct tract degeneration was absent except for a few degenerated fibers of Goll's column. Sections of the medulla in its lower part showed very minute capillary hemorrhages chiefly in the gray matter. The brain was not examined.

CASE III.—C. M., a white female, aged fifty years, was admitted to the Philadelphia Hospital in the service of Dr. Spiller complaining of weakness of the lower limbs. Syphilis was denied. Three months previous to admission patient first developed some pain about the left shoulder which resisted all treatment. About two months later both legs began to grow weak, this gradually progressed until one week before admission when she was unable to walk or stand without assistance.

Physical Examination: The patient is much emaciated. The pupils are equal and react normally to light and accommodation. Movements of muscles of the face, tongue, larynx, and pharynx are normal. The power of the upper extremities is equal and normal. Triceps and biceps jerks are equal and normal. Both lower extremities are completely paralyzed and there is extreme contracture at the knees and hips. From time to time there are violent jerking movements at the knees. Patellar tendon reflex is normal on the left, but slightly decreased on the right. Achilles reflexes are equal and normal. Clonus is absent. There is distinct Babinski signs on both sides. Sensation for pin prick and for touch is lost over both lower extremities and abdomen as far as the fifth interspace, at this point pain and tactile sensations are impaired but not lost. Sensation for heat and cold was not tested. The vertebral column although prominent on account of emaciation showed no deformity.

Macroscopic examination showed tuberculous caries of the second thoracic vertebra and an external tuberculous pachymeningitis beginning one quarter of an inch above the exit of the second thoracic root and extending one quarter of an inch below the exit of the third thoracic root, therefore involving the whole of the intradural portion of the third thoracic root from its exit from the cord to its exit through the dura. Microscopic examination of section taken from the third thoracic segment showed no change in contour. When stained by the hemalum and fuchsin method the pia seemed normal, but there was a slight mononuclear cellular infiltration of the adventitia of some of the pial vessels. In the white matter the walls of the bloodvessels were thickened and there was a very slight perivascular infiltration of mononuclear cells and an increase of the glia cells. There were no distinct areas of softening.

In the gray matter there were many mononuclear cells diffusely scattered. By the Nissl stain the ganglion cells were few in number and those remaining were much degenerated. With the Weigert method the medullary sheaths were swollen and many axis cylinders had disappeared. By the Marchi method nearly all the fibers of the white matter were degenerated, but a few scattered normal fibers still remained on the lateral columns near the periphery. Above and below the site of compression distinct tract degeneration was absent, though a few fibers were found staining very darkly in the pyramidal tracts and in the posterior columns by the Marchi stain.

In cases of tuberculous caries infection is generally carried to one of the vertebræ from some other focus within the body such as the lung, and a chronic osteomyelitis or periostitis ensues. This may go on to necrotic softening, and then by purely mechanical means the adjacent vertebræ above and below become approximated producing the well-known deformity of Pott's disease. In some instances this deformity may be sufficient to cause direct pressure upon the cord, but as Kraske has shown, it is uncommon; he having found it in only 6 of his 58 cases. Occasionally a subperiosteal abscess may exert pressure; Oppenheim and others have even seen cases in which spicules of necrotic bone encroached upon the cord. In the great majority of cases however, infection extends to the external lateral and posterior surface of the dura setting up a chronic inflammation which produces great thickening with epidural exudates and fungoid proliferations. Fickler in 20 cases found compression caused by dislocation of a vertebra in 9 per cent.; by abscess formation, in 17 per cent.; while pachymeningitis externa was responsible for 73 per cent. Infection of the dura nearly always arises by direct extension from a focus in one of the vertebræ, yet Schlesinger, Henneberg, and Rossi, have each reported a very uncommon condition in which there was tuberculous pachymeningitis without caries of the vertebræ.

A similar case has recently been under the care of Dr. Spiller, and Dr. Frazier, the notes of which are as follows:

CASE IV.—H. B., a male, white, was admitted to the University Hospital, April 10, 1909, in the service of Dr. Spiller.

The patient's father, grandfather, one aunt, and one sister died of tuberculosis. Six years ago the patient had scarlet fever which was followed by paralysis of all four extremities lasting two months and then he entirely recovered. Seven months previous to admission he first developed pain in the region of the sixth dorsal vertebra. Pain was present on movement and on jarring of the spinal column, and continued in spite of treatment. About five weeks ago he noticed his legs were growing weak and that he staggered when walking. Occasionally he had cramp-like pains in the calf muscles of both legs. This condition increased gradually until two weeks ago when he was not able to walk at all. Pain still continued in the lower legs. Patient cannot control bowels and can retain urine only a short time, and is troubled with distention of the abdomen. He states that for the last four weeks he could not feel distinctly below the hips.

The following notes were dictated by Dr. Spiller: Patient is completely paralyzed in both extremities, slight movement of drawing upward of the left lower leg seems to be from muscles of the trunk. Limbs are not wasted. Patella and Achilles reflexes are about normal on each side. No ankle clonus on either side. Dr. Willard, Jr., noticed bilateral ankle clonus three days ago, before patient entered hospital. Tactile sensation seems to be preserved in all parts of the body except possibly over the front and outer side of the thighs. Here he sometimes answers incorrectly. Pain, heat, and cold sensations are lost or greatly impaired over both lower limbs and trunk anteriorly and posteriorly to a line three inches below the nipple. Area of disturbed sensation shades off gradually into an area of normal sensation. Babinski sign is typical on each side. Testing him with pin prick, or heat, and cold, causes at times either lower limb to be forcibly drawn upward. The upper thoracic vertebræ are distinctly arched, but there is no sharp kyphosis, only gradual arching. Von Pirquet tuberculin test was positive, twenty-four hours. Urine was negative. Blood count was: hemoglobin, 48 per cent.; red blood corpuscles, 4,880,000; leukocytes, 11,900.

Additional notes dictated by Dr. Spiller, May 19, 1909: Both legs are completely paralyzed. There is considerable desquamation of the skin of the feet and legs below the knee. There is involuntary drawing upward of lower limbs at hips and knees. Has imperfect control over bladder, cannot hold urine; no control over rectum. Only pain he has now is at lower costal margin, and there is no atrophy of any part of the body or even of the lower limbs. Lower limbs are very spastic. Knee jerks are exaggerated on both sides. Patellar clonus present on both sides. Achilles jerks are exaggerated on both sides but clonus is not obtained on either side on account of spasticity. Any irritation of the limbs or trunk causes limbs to be drawn forcibly upward. Touch, pain, heat, and cold sensations are lost in the lower limbs and trunk anteriorly and posteriorly to a line drawn around the body from the eleventh and twelfth thoracic vertebræ to the sixth interspace on the left side in the nipple line, and seventh interspace on the right side in the nipple line. This line is very sharp for touch but not so sharp for pain and temperature sensations. There is no involvement of the upper limbs. Prominence of vertebræ is in the midthoracic region but no actual displacement.

On June 24, Dr. Frazier removed the fourth, fifth, and sixth dorsal spinous processes. During the operation the following notes were dictated. Coming out between the transverse processes of the fifth and sixth dorsal vertebræ was found a mass of tissue about the size of a marble presenting the appearance of granulation tissue. Upon exposure of the cord it was found free from involvement, the tumor tissue seemed to come out from either right side of the body of the vertebra or from the transverse process of the fifth dorsal. All tumor tissue that could be was removed with scissors and curette. There was evidently compression of the cord at the site of injury which was relieved by removal of the laminæ. Tumor did not make pressure over the dorsal but over the lateral aspect of the cord. Recovery after operation was good.

The pathological report was as follows: The specimen consists of laminæ and small portion of spinal meninges with nerve cord about three inches long. Microscopically, soft tissue removed from laminæ shows simple areolar tissue, the fibrous elements of which are somewhat thickened and in places infiltrated with small round cells, also a few degenerated muscle fibers are seen and many typical tubercles were found with epithelioid cells and giant cells.

On October 11 examination of the patient showed there was no return of power in the lower extremities. Sensation to pain and touch is lost below the fifth interspace on the right side anteriorly and the sixth space anteriorly on the left side. Patient complains of sharp pain in the region of the right nipple on attempting to raise head from bed.

In attempting to explain the intraspinal lesions of Pott's disease it must be borne in mind that there are two cardinal factors each of which originate outside the dura, namely, pure mechanical compression and infection. Charcot and Michaud believed that degeneration of the spinal cord was caused by infection extending into the substance of the cord setting up a secondary myelitis;

on the other hand, Kahler was able to show by compressing the spinal cords of animals with non-toxic substances that lesions similar to those following caries of the vertebræ resulted. Later Leyden, recognizing the possibilities of infection, declared that compression in itself was an important factor. Strümpell strongly opposed the myelitic theory explaining the alterations in the cord as the result of œdema stoppage of the lymph stream, and anemia, secondary to pure compression; Schmaus was inclined to accept Strümpell's view but modified it somewhat by adding that there was a collateral inflammatory œdema which could be attributed to toxic influences; more recently, however, in an article upon myelitis the same author has expressed the opinion that constant prolonged pressure upon the cord may lead not to stasis œdema but to true secondary inflammation, that is, myelitis.

The first three cases which I have described presented swelling and destruction of axis cylinders, proliferation of the glia, round-cell infiltration, sclerosis of the bloodvessels, perivascular infiltration, necrotic softening of the central gray matter, and compound granular cells at the level of greatest compression of the spinal cord. In Case I there was an internal hydrocephalus affecting only the lateral ventricles; the formina, however, were not occluded, but the cells of the choroid plexus were decidedly diseased; it is conceivable, therefore, that as a result of perverted function of diseased cells there may have been an increase of secretion sufficient to cause this distention. But it is also possible that hydrocephalus may have been the result of compression of the cord, and that if an ophthalmological examination had been made choked disk would have been found. Choked disk has not been seen frequently in Pott's disease, yet it has been observed in spinal tumor by Bailey. At other levels of the spinal cord where compression seemed to play no part there were definite though very moderate signs of vascular disease, consequently it might seem as if there was some other element responsible as well as compression.

Spiller in an article on Pott's disease refers to a form of simple meningomyelitis without giant cells or miliary tubercles, occurring particularly when Muller's fluid had been used for preservation, he also quotes Oppenheim who had called attention to a similar type of myelitis in persons dying of generalized tuberculosis. In my cases there were no specific signs of tuberculosis found within the dura, yet the diagnosis of Pott's disease is evident, and it does not seem probable that the existence of myelitis would be disputed.

The symptoms of cord involvement in Pott's disease vary considerably in their mode of onset, but after having once begun they frequently progress rapidly. Usually the onset is chronic, the signs of paralysis requiring some months to reach their height. Occasionally paraplegia develops in the course of two or three weeks. Very rarely, however, there is an instantaneous onset, and, moreover, in such cases there is usually a history of trauma, or some undue strain upon the diseased vertebral column immediately preceding the initial paresis or paralysis, which causes sudden displacement of one of the vertebræ and thus brings about rapid compression of the spinal cord. Gowers describes a child with angular deformity of the spine, who, while walking across the room, suddenly fell to the floor and after being lifted up was paralyzed in both legs. In Case IV not only was deformity absent, but, as far as could be determined at operation, the vertebræ were entirely normal. Deformity was entirely lacking in the other three cases at the time of the onset of paraplegia, yet in Case I and Case II the development of paralysis could not have been more rapid. In the first case, motor and sensory paralysis of one lower extremity was said to have begun instantaneously, the patient's right leg suddenly giving way under him while walking, and one week later the left leg was similarly affected. The second patient developed paralysis of both lower extremities in an equally rapid manner. Truly an apoplectiform onset in the strictest sense of the term.

After searching the literature upon this subject I have been able to find only two cases described in which paralysis developed instantaneously without deformity of the vertebræ. One case with necropsy reported by Raymond seventeen years ago seems to have been entirely overlooked, another without necropsy was described by Martineck during the past year. Raymond's case is as follows: A young girl developed complete motor paralysis of both upper extremities and incomplete motor paralysis of the lower limbs within a few hours. Laminectomy was performed and the pia was found to be thickened and the cord much congested, but without evidence of compression. The patient died on the same day and at the autopsy tuberculous caries of the third cervical vertebra and abscess formation was found. Microscopic examination showed inflammation and hemorrhage in the gray matter, sclerosis of the bloodvessels and cellular infiltration of the pia, while the white matter was much less involved, yet there were no specific lesions of tuberculosis within the dura.

Martineck's case was that of an adult male, who had been complaining for some months of severe pain. The patient went to bed feeling generally weak, the next morning there was paresis of both legs, by the afternoon there was paralysis of both lower extremities. When the patient was seen at the hospital the following day there was almost complete motor paralysis and involvement of the bladder, still the vertebral column showed no abnormality. There was neither tenderness nor pain on movement. It was not until eight days after paralysis had developed that an angular deformity was discovered extending from the eighth to the eleventh dorsal vertebræ. *X*-ray examination showed abnormality in the position of the ninth dorsal vertebra.

When the term myelitis is used in its strictest sense to mean inflammation of the spinal cord, Russel believes that it is a rare disease, and attributes much of which we speak of as myelitis to softening consequent upon vascular occlusion, in which inflammation plays no part, unless it is some inflammatory process in connection with the walls of the bloodvessels which tends to thrombosis and later softening. Douglas Singer also refers the majority of cases of myelitis to primary thrombosis.

It is well known but perhaps not thoroughly appreciated, that in cases of syphilis occlusion of some of the smaller spinal vessels produce sudden weakness and paralysis; in fact, sudden onset of paralysis is common in syphilitics. Spiller, in an article upon syphilitic acute anterior poliomyelitis due to thrombosis, emphasizes the importance of considering the rapid development of paralysis and says "spinal thrombosis is not a rare condition and is probably the cause of most of the apoplectiform palsies that occur in myelitis." In my case, syphilis, dislocation of vertebræ and trauma can all be excluded with certainty, yet a traumatic cause, if this expression is permissible, was present, that is to say compression. As has been already pointed out, compression of the spinal cord even when slight, can in itself cause œdema and inflammation with thickening of the bloodvessels within the cord, in other words, a fertile soil for thrombosis. Epidural inflammation and fungoid proliferations increase very gradually up to a certain point without evident disturbance in the functions of the cord unless it be pain; but when this point has been reached a very minute sudden increase may be sufficient to give signs of rapidly developing weakness or palsies, on the other hand, it is possible that it is just at this stage of the disease that thrombosis develops. Whether or not in my cases the initial palsies were caused by sudden increase of pressure with thrombosis, or to increased pressure without thrombosis, or to the changes which were found within the cord, is of course, problematical.

Raymond was inclined to look upon his case as one of hematomyelia, also Huber and Vorkarstner in discussing Martineck's case suggested the possibility of there having been a hemorrhage within the gray matter of the cord in addition to compression. Spontaneous hemorrhage in the spinal cord is exceedingly rare. Minor who has studied this question minutely, says that among all the cases which he has observed (other than Pott's disease) hemorrhage occurred only twice without preceding trauma. In an anatomical sense it is always difficult to make a sharp distinction between primary hemorrhage with subsequent myelitis from primary hemorrhagic myelitis and in many instances absolutely impossible.

In Cases I and II it was impossible to prove conclusively that thrombosis had taken place, nevertheless on account of marked thickening of the bloodvessels throughout the cross section of the cord, together with extensive central softening and similarity of the onset of paralysis to cases of primary myelitis in which thrombosis has been proved, it seems more than probable that thrombosis may have occurred. At all events, instantaneous paralysis in Cases I and II and the more slowly developing paraplegia in Cases III and IV was the clinical expression of an intense myelitis and was beyond all doubt the direct result of compression of the spinal cord by means of tuberculous external pachymeningitis.

In determining the upper limits of a focal lesion causing compression of the cord, particularly when situated in the dorsal region, there is always more or less uncertainty. The most important single sign is the character and extent of sensory loss of function. Certain authors rely upon the level of hyperesthesia so frequently found just above the level of the total loss of sensation. In an article on spinal tumors Bailey maintains that the upper limits of incomplete loss of sensation is probably the most accurate guide in determining the exact level of the lesion. In two of my cases the lesions were too diffuse to draw accurate conclusions, but in Cases III and IV the limits of hypesthesia of the skin indicated quite accurately the segment of the cord which was involved.

The diagnosis of Pott's disease when deformity of the spine is present is generally easy, but, as Moussaud has pointed out, without this characteristic sign an accurate diagnosis may be exceedingly difficult or impossible. Fortunately, cases of this kind are uncommon, yet unmistakable evidence of caries is by no means always present. Nevertheless by a careful consideration of the clinical history, bearing in mind the similarity of the onset of paralysis in certain cases of Pott's disease with that of other form of myelitis particularly in syphilitics, and by use of *x*-ray examinations, tuberculin test, the Wassermann and Noguchi reactions, and lumbar puncture, in a certain number of cases at least, an accurate conclusion can be reached. If in a given case it can be proven that the bones of the vertebral column are not involved and the initial paralysis did not begin suddenly operation should be seriously considered. In such cases it is probable that the symptoms are caused by slowly increasing pressure upon the cord by a dry inflammatory exudate with a minimum amount of pus, or pus may be entirely absent as in Case III; therefore, if operation is done early and pressure removed recovery might be possible. Recovery does not seem to occur without operation. On the other hand, an apoplectiform onset of paralysis would indicate serious intraspinal lesions and operative interference might be of no advantage, and yet it is probable that in these cases also, compression is the primary etiological factor; if, therefore, it can be removed by operation, the injured cord will have better opportunity to recover its normal function, although in Case IV operation was of little benefit.

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THE PATHOLOGICAL FINDINGS IN THE PARATHYROIDS IN A CASE OF INFANTILE TETANY.

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In the year 1880, the Swedish anatomist, Sandström, first described the parathyroids in man and also in the horse, cattle, cat, dog, and rabbit. In the same year, Nathan Weiss gave the first description of three cases of tetany after thyroidectomy; the condition was at that time called tetania strumipriva. Today, after thirty years, it is generally considered that such tetany, whether operative or experimental, is due not to the loss of the thyroid, but to the removal of the parathyroids (Vassale and Generali, Biedl, Pineles, Erdheim). In other words, the condition is not a tetania strumipriva, of the same origin as myxœdema and cachexia strumipriva, but a tetania *parathyreopriva*.

In regard to the so-called medical or spontaneous tetanies, however, it is at present still merely an hypothesis[1] that they are due to insufficiency of the parathyroid. Among such spontaneous tetanies

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[1] For opposition to this theory see Berkeley, Forsyth, Kassowitz, and Thompson.